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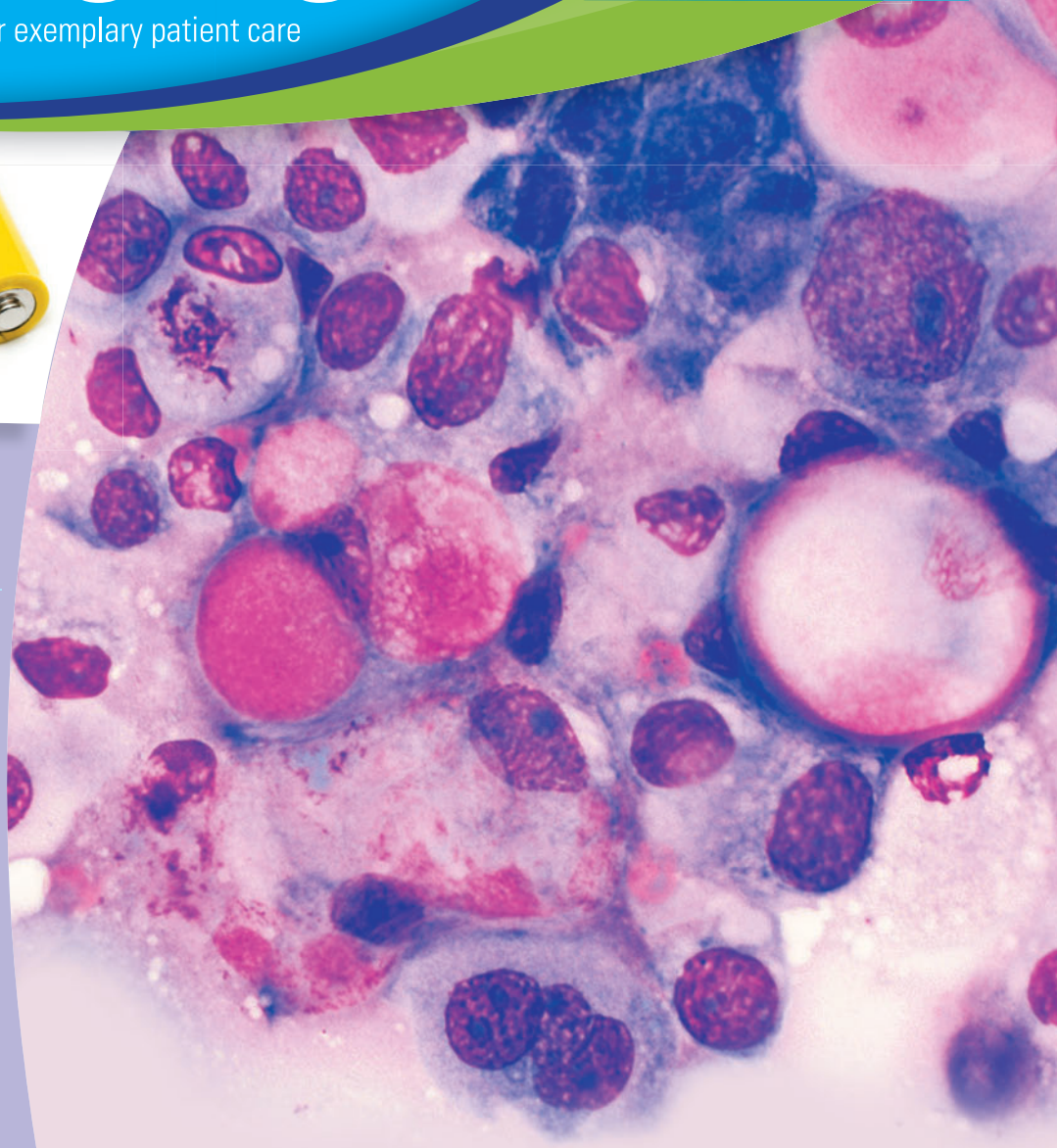

Transitional cell CARCINOMA

Get an entire clinical team's point of view on the best way to diagnose and treat this urinary tract neoplasia in dogs. p327

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Anesthesia reboot: Erase these *myths* and *misconceptions*

Veterinary anesthesiologist Dr. Ann Weil provides five important pointers on anesthetizing cats and dogs.

By Ann Weil, DVM, DACVAA

Are you up-to-date on the best anesthetic practices to use in your patients? You may be holding on to important misconceptions, including the following:

1. It is true that most opioids can cause hyperthermia in cats. A recent study has shown a strong association of hyperthermia with hydromorphone, especially when ketamine is used concurrently.[1] My solution: Administer a microdose of medetomidine (1 µg/kg) postoperatively in cats that have received hydromorphone and ketamine.
2. Misconceptions abound in terms of breed "sensitivities" to anesthetic drugs. The only documented sensitivity is associated with greyhounds and barbiturates. Their livers cannot process these drugs in the same manner as other dogs. However, since barbiturates are rarely used anymore, this problem is almost obsolete.
3. Excessive bradycardia from dexmedetomidine should be handled by reversing the drug with atipamezole, not by using an anticholinergic such as atropine or glycopyrrolate. Partial reversal can be done with a half dose of atipamezole.
4. If you are giving injectable combinations of anesthetic drugs, keep in mind that hypoxemia and hypercapnia are likely. Thus, be sure to use an oxygen mask in these patients.

5. Stop using the old-school 10 ml/kg/hr anesthetic fluid rate as a guide. The 2013 American Animal Hospital Association and American Association of Feline Practitioners fluid therapy guidelines advise new initial fluid rates: 5 ml/kg/hr in dogs and 3 ml/kg/hr in cats (*see* **dvm360.com/FluidGuidelines**).

REFERENCE

1. Posner LP, Gleed RD, Erb HN, et al. Post-anesthetic hyperthermia in cats. *Vet Anaesth Analg* 2007;34(1):40-47.

CVC

Pain control in cats

To hear Dr. Weil discuss the ever-evolving role of butorphanol in treating pain in cats, scan the QR code below or visit **dvm360.com /CVC14Weil**.

WHEN TREATING SKIN INFECTIONS, VETERINARIANS—AND PET OWNERS—ARE CHOOSING BEST MEDICINE OVER LOWEST COST

The cost of their pets' healthcare can be a concern for clients—and a discussion veterinarians would sometimes just as soon avoid. This can be the case when treating skin infections, one of the most common health issues bringing clients and their pets into the veterinarian's office.[1] However, a new study demonstrates that clients, including owners of dogs up to 135 pounds, may be less concerned about cost than practitioners often believe.

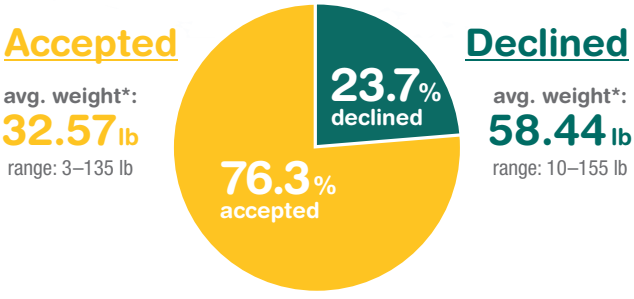
Veterinarians are familiar with CONVENIA® (cefovecin sodium), an injectable antimicrobial indicated to treat common bacterial skin infections in dogs and cats; however, they may be reluctant to offer it as a first-line treatment because of concerns their clients may consider it too costly. Zoetis recently conducted a study to determine if, in fact, cost was a deterrent to treatment with CONVENIA.

"There is a perception dog owners will not accept the cost of cefovecin sodium compared to less expensive oral medications requiring repeated administration," said Andrea Wright, DVM, MVSc, MBA, associate director, Outcomes Research in the Companion Animal Division at Zoetis.[2]

Thirty veterinarians, who are members of Companion Animal Practices North America (CAPNA™) and are located in 22 different geographical areas of the United States, were asked to offer CONVENIA as a first treatment for appropriate cases. The date of examination, dog weight, and owner acceptance or decline were recorded over two months. The pricing was consistent across all clinics: owners were charged using the weight of the dog multiplied by the clinic's cost of CONVENIA per lb, plus a flat margin.

"The most surprising result was that such a high number of clients, 76.3 percent, accepted treatment with CONVENIA even though the cost was much greater than oral antibiotics requiring repeated owner administration," Dr. Wright said. In fact, the cost of one-time treatment ranged from three to 17 times more than the oral alternatives.

Pet owner acceptance of injectable cefovecin sodium as first line of treatment



One Study Participant's Experience

Todd Rezac, DVM, of Deer Creek Animal Hospital in Littleton, Colorado, participated in the study and was pleased with the results: "When given a choice between two weeks of orally administered medication and one injection at a higher fee, my clients accepted the injection 100 percent of the time." Dr. Rezac noted the response was the same with owners of big dogs and small ones. "I used CONVENIA on dogs weighing 100 pounds or more, and none of my clients refused because of cost."

Dr. Rezac added, "As clinicians, it is our job to offer the best medicine and when treating skin disease, in my view, CONVENIA is the superior product."

He went on to note, "Not only does one injection provide assurance that the full course of treatment has been administered, it removes the client's need to give daily medication and helps alleviate the patient's discomfort more quickly. That's what I tell my clients and let them make the decision."

IMPORTANT SAFETY INFORMATION:
People with known hypersensitivity to penicillin or cephalosporins should avoid exposure to CONVENIA. Do not use in dogs or cats with a history of allergic reactions to penicillins or cephalosporins. Side effects for both dogs and cats include vomiting, diarrhea, decreased appetite/anorexia and lethargy. For more information, please see Brief Summary of full Prescribing Information on page 321.

convenia® cefovecin sodium

[1]Banfield State-of-Pet Health 2014 report.
[2]Wright A, Russo S, Di Franco B, Amodie D, Gasper S. Convenia Client Acceptance Experience Trial. Data on file, 2013, Zoetis Inc.
*The difference in mean weights between accepted and declined was statistically significant at $p<0.0001$.

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CASE STUDY
The cat that suffered an Addisonian crisis

Overview

The authors of this case report describe a 4-year-old spayed female British shorthaired cat that presented with severe weakness, bradycardia (120 beats/min), hypovolemia, and hypothermia (99 F [37.2 C]). The cat's systolic blood pressure was measured and was undetectable.

The owners reported that the cat had been polyuric and polydipsic beginning four weeks earlier but had become progressively weaker and had stopped eating or drinking one week prior to presentation.

Diagnostic testing. The results of a complete blood count showed lack of a stress leukogram. The following serum chemistry profile abnormalities were found (reference ranges in parentheses):

- pH = 7.14 (7.31 to 7.39)
- Bicarbonate (mmol/L) = 10.4 (15.5 to 23.9)
- Base excess = -19 (-10.12 to 1.2)
- Sodium (mmol/L) = 121 (141 to 150)
- Potassium (mmol/L) = 8 (3.6 to 4.8)
- Chloride (mmol/L) = 90.8 (110 to 125)
- BUN (mg/dl) = 62.5 (20 to 30)
- Creatinine (mg/dl) = 6.7 (0 to 1.9)
- Phosphorus (mg/dl) = 11.5 (2.5 to 5.9)

The blood glucose concentration was also noted to be at the low end of the reference range. A thoracic radiographic examination revealed microcardia, a narrow caudal vena cava, and pulmonary hypoperfusion. An abdominal ultrasonographic examination revealed a small left adrenal gland; the right adrenal gland could not be visualized.

Treatment. Intravenous crystalloids (0.9% sodium chloride) and hydroxyethyl starch were given as well as sodium bicarbonate to correct the meta-

GETTY IMAGES/ANNA LURYE

bolic acidosis. Rewarming measures were also taken. Dextrose was added to the fluids, and fluid content was adjusted according to blood gas analysis results. An ACTH stimulation test was performed at the initiation of therapy.

After aggressive therapy and monitoring (continuous echocardiography and blood gas analyses every three hours) over 12 hours, the cat improved and vital parameters normalized. An oral dose of 0.025 mg fludrocortisone given twice daily and 0.3 mg/kg prednisolone given once daily was begun within 24 hours of presentation. Over the next few days the cat continued to improve, and its clinical signs resolved.

Definitive diagnosis. The ACTH stimulation test confirmed a diagnosis of hypoadrenocorticism with both cortisol and aldosterone concentrations not detectable at baseline and after stimulation. At the one-week follow-up visit, mild electrolyte abnormalities were still present, but the results of a physical examination were normal. The prednisolone was discontinued at this time, and the fludrocortisone was continued as before. Two weeks later, the polyuria/polydipsia returned and the prednisolone was reinstituted. The clinical signs resolved, and the cat was maintained on a 0.2 mg/kg once a day prednisolone dose in conjunction with the fludrocortisone.

Take-home message

Addison's disease is rare in cats, and the cause remains unclear. The clinical presentation and clinicopathologic abnormalities in this case were similar to what we would expect to see in dogs, except for the severe metabolic acidosis. With appropriate diagnosis and treatment, the long-term prognosis in cats with hypoadrenocorticism is favorable. VM

Sicken J, Neiger R. Addisonian crisis and severe acidosis in a cat: a case of feline hypoadrenocorticism. *J Feline Med Surg* 2013;15:941-944.

These "Journal Scan" summaries were contributed by Jennifer L. Garcia, DVM, DACVIM, a veterinary internal medicine specialist at Sugar Land Veterinary Specialists in Houston, Texas.

Read more summaries at **dvm360.com/JournalScan**.

AA toxicosis: *Alkaline battery* exposure in a dog

A look at the case management of a 1-year-old Australian shepherd who presented to a veterinary clinic for evaluation after chewing on two AA alkaline batteries.

By Camille DeClementi, VMD, DABT, DABVT

A 1-year-old 26-lb (11.8-kg) spayed female Australian shepherd was presented to a veterinary clinic for evaluation one hour after chewing on two AA alkaline batteries.

History

At home, the owner found two AA alkaline batteries with two small puncture holes in them. The owner noticed a red blotch on the dog's tongue and brought the dog to the clinic.

Physical examination

On physical examination, the dog was bright, alert, and responsive. A mild superficial abrasion was noted along the left buccal surface of the tongue. No other significant changes were noted, and the physical examination was otherwise unremarkable.

Initial treatment

Oral irritation was already present at the time of the initial examination, so the dog was administered medications to protect the gastrointestinal (GI) tract. These included sucralfate (500 mg mixed in water until a slurry forms and then given orally t.i.d. for five days) and famotidine (10 mg orally once daily for five days).

Since damage to the mucosa can be delayed and the full extent of the damage may not be recognizable until about 12 hours after the exposure,[1] the owner was instructed to monitor the dog at home for worsening lesions in the mouth, hypersalivation, lethargy, anorexia, and vomiting.

Further treatment

About 18 hours after the exposure, the dog became lethargic, developed mild hypersalivation and anorexia, and was again presented to the veterinary clinic. The physical examination revealed moderate ulceration of the buccal mucosa and tongue. The dog's temperature was normal, and abdominal palpation did not seem to elicit pain. Results from a complete blood count and serum chemistry profile were normal. Tramadol (50 mg orally t.i.d. for five days) and amoxicillin-clavulanic acid (125 mg orally b.i.d. for seven days) were administered.

The dog was hospitalized for monitoring. Within four hours of hospitalization, the patient began eating small amounts of critical care canned dog food.

The dog was discharged 12 hours after admittance.

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The owner was instructed to continue giving all medications (sucralfate, famotidine, tramadol, amoxicillin-clavulanic acid) for the labeled time frames and to only feed the dog canned food until the lesions in its mouth resolved. The dog was fully recovered seven days after the exposure.

Discussion

Dry cell batteries contain an electrolyte solution or gel of either potassium hydroxide or sodium hydroxide, both of which are alkaline. Chemically, alkaline materials generate an aqueous solution in which there are more hydroxyl (OH-) ions than hydrogen (H+) ions.[2] Alkaline materials can cause corrosive injury on contact with tissues through a process called *liquefaction necrosis,* which includes protein dissolution, collagen destruction, fat saponification, and cell membrane emulsification. During liquefaction necrosis, the tissues soften and allow the alkali to penetrate deeply into the tissues.[1] Exposure to an alkaline material causes little to no pain initially, unlike exposure to an acid. Thus, patients are not deterred from accidental exposure.

In cases in which a battery is chewed and punctured, the alkaline material may leak out and cause localized tissue damage. If the battery was not swallowed, the damage is most likely to occur in the mouth, but it is also possible that there will be damage in the esophagus and farther down the GI tract. The esophagus is most at risk since it does not produce protective secretions that dilute the toxin like the mouth

and stomach do. Lesions in the esophagus may lead to perforation or, when healed, scar formation and contracture, leading to long-term esophageal stricture.[1]

If the battery has been ingested, in addition to the above concerns, it may also pose a foreign-body-obstruction threat, or if the battery remains in the stomach long enough for the casing to break down, heavy metals such as zinc or lead may be released and lead to toxicosis.

Clinical signs of irritation of the lips or tongue or in the mouth may occur within a few hours of exposure to a chewed battery, and ulceration may take up to 12 hours to fully develop.[1] The patient may also exhibit the clinical signs of lethargy, hypersalivation, dysphagia, anorexia, vomiting (potentially with blood), abdominal discomfort, hyperthermia, and melena.[1] Hyperthermia occurs, possibly because of corrosive tissue damage and inflammation from the alkali. Affected animals often show mild to moderate leukocytosis.

If the battery that was ingested is a button or disc battery, there are additional risks. Button batteries are found in many electronic devices including hearing aids, games, watches, calculators, and greeting cards. As described above, alkaline liquid can also seep from damaged disc batteries. In addition, there is the added risk of injury to adjacent tissues from the current flowing between the cathode and anode, which results in electrolysis of endogenous salt and the production of corrosive sodium hydroxide[2] if the battery becomes lodged in the GI tract, particularly the esophagus. Thus, significant corrosive damage, including esophageal burns, necrosis, and perforation, can occur even if the button battery does not leak.[1]

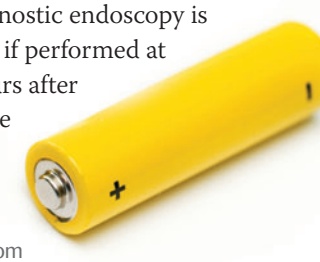
Differential diagnoses

Mucosal burns can also be caused by thermal or electrical injury or due to exposure to other corrosive materials, such as acids, disinfectants, cationic-containing detergents or potpourri, automatic dishwasher detergent, formaldehyde, concentrated bleach (sodium hypochlorite), and phenols.

Diagnosis

Diagnosis is based on a history of exposure and the consequent development of any of the clinical signs listed above. Since batteries have metal casings, radiography can be helpful in determining if any portion of the battery was swallowed and, if so, the location of the battery in the GI tract. In cases of button battery ingestion, radiography is particularly helpful in determining if the battery has become lodged in the esophagus.

Endoscopic examination of the oropharynx, esophagus, and stomach by using a flexible endoscope is recommended to determine if ulceration has occurred (since esophageal ulceration may be present even if no ulcers are present in the mouth) and to determine the severity of ulceration.[1] Diagnostic endoscopy is most useful if performed at least 12 hours after the exposure because the



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See brief summary on page 324.

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development of the ulceration may be delayed, but it should be performed within 24 hours of the exposure. If a battery is identified radiographically in the esophagus or stomach, therapeutic endoscopy to remove the battery may be indicated.

Treatment

Patients that have ingested corrosive materials should be given a small amount of milk or water immediately to dilute the corrosive material. (In this case, the window of opportunity for dilution had passed by the time the patient was presented to the clinic.) The recommended dose is 2 to 6 ml/kg orally, which is about 3 to 9 tbsp in a 50-lb dog.[3] If a button battery was ingested, giving serial doses of water (20 ml every 15 minutes) may delay and lessen the severity of the lesions.[3]

If the battery is leaking, emesis is contraindicated to prevent additional exposure of the esophagus to the alkaline corrosive material. Activated charcoal is also contraindicated since it is unlikely to bind to the corrosive material, may make visualization of ulceration difficult, and could be released into the abdominal cavity if GI perforation develops.[3]

Once a button battery has moved into the stomach, if it is not large compared with the size of the patient, it will likely continue to pass through the GI tract and be expelled in the feces. A button battery lodged in the esophagus should be removed through endoscopy immediately since marked esophageal mucosal necrosis was observed in dogs after as little as 15 minutes of contact with a 3-volt disc battery.[1]

Bulking agents, such as wheat bread, psyllium, or canned pumpkin, may be helpful in moving small button batteries or battery pieces through the GI tract.[3] Sucralfate (0.5 to 1 g orally as a slurry t.i.d.) should be given to patients that develop mucosal irritation or ulceration, in addition to giving an H_2 blocker (*e.g.* famotidine 0.5 to 1 mg/kg orally daily to b.i.d.) or a proton pump inhibitor *(e.g.* omeprazole 0.5 to 1 mg/kg orally once a day).[4] These medications should be given until the clinical signs have resolved, often in seven to 10 days or longer. Patients with ulceration should also be given a broad-spectrum antibiotic to combat secondary infection.

Anorectic patients may need nutritional and fluid support. Opioid or opioid-like medications, such as tramadol (2 to 5 mg/kg orally two to four times a day), a fentanyl patch, or buprenorphine (0.005 to 0.02 mg/kg two to four times a day) can be given to lessen the pain of ulceration.[4]

Monitoring

Monitor patients for 24 hours for clinical signs of GI irritation or ulceration. If ulceration develops, monitor the patient's complete blood count and body temperature. Perform endoscopy of the stomach or esophagus if needed to observe the extent of mucosal damage or to remove a battery seen on radiographs.

Summary

Alkaline battery exposure in dogs can cause corrosive injury and GI ulceration, especially in the mouth and esophagus. Esophageal ulceration can lead to perforation and stricture formation. Treatment measures may include immediate dilution with milk or water; the administration of GI protectants, pain medications, broad-spectrum antibiotics, liquid food, and supportive care; and the removal of the battery or pieces of the battery through endoscopy or surgery. VM

REFERENCES

1. Gwaltney-Brant S. Batteries. In: Plumlee KH, ed. *Clinical veterinary toxicology*. St. Louis: Mosby Elsevier, 2004;140-142.

2. Litovitz T. Button batteries. In: Ford MD, Delaney KA, Ling LJ, et al., eds. *Clinical toxicology*. Philadelphia: WB Saunders Co, 2001;1027-1031.

3. DeClementi C. Prevention and treatment of poisoning. In: Gupta RC, ed. *Veterinary toxicology: basic and clinical principles*. London: Elsevier, 2012;1364-1368.

4. Plumb DC. *Plumb's veterinary drug handbook*. 5th ed. Ames, Iowa: Blackwell Publishing, 2005; 321, 573, 722, 774.

Clinical Rounds CE

Transitional cell carcinoma

Thousands of dogs develop this form of cancer every year. Follow along as these clinicians delve in the particulars of a case in a senior Chihuahua—and see how you can best prolong survival in the next patient you diagnose with this form of neoplasia.

Transitional cell carcinoma (TCC) is the most common urinary tract cancer in dogs, affecting thousands each year.[1-4] Risk factors for developing TCC are multiple and include obesity, female sex, exposure to older-generation flea control products, and exposure to herbicides and pesticides.[2,5-8]

Scottish terriers, Shetland sheepdogs, beagles, wire-haired fox terriers, and West Highland white terriers have a predilection to develop TCC.[2,5-8] The Scottish terrier is the most at-risk breed for developing this tumor—an odds ratio of 18.09.[2] Additionally, obese female dogs exposed to insecticides are 28 times more likely to develop TCC than normal weight dogs.[6,8] Interestingly, consuming vegetables at least three times a week in addition to a normal diet seems to reduce the risk of developing TCC in dogs.[9]

A "field effect" has been proposed for these tumors and refers to the multifocal changes that occur in the bladder as a result of exposure to carcinogens in the urine that cause malignant changes throughout the bladder epithelium. Metastasis to the regional lymph nodes and the lungs is noted in about one of six cases at the time of diagnosis, but it is more common later in the course of disease, with about half of dogs developing metastasis to the lymph nodes, lungs, other abdominal organs, or bones at the time of death.[5,10,11]

Dogs usually present with lower urinary tract signs (*e.g.* hematuria, stranguria, pollakiuria, dysuria), and many have concurrent urinary tract infections (UTIs) that may initially delay the diagnosis. Resolving the infection may temporarily alleviate the clinical signs, and patients often present with a several-month history of lower urinary tract signs. A middle-aged dog with its first UTI should prompt imaging of the bladder to rule out bladder stones or a bladder mass.

CASE PRESENTATION

An about 16-year-old spayed female Chihuahua (5.5 lb [2.5 kg]) presented to the University of Tennessee Veterinary Medical Center for evaluation of a suspected bladder mass found on ultrasonographic examination by the primary care veterinarian. The owners reported that the dog had a history of hematuria for about three months before presentation.

Diagnostic testing

Staging tests included a complete physical examination, a complete blood count, a

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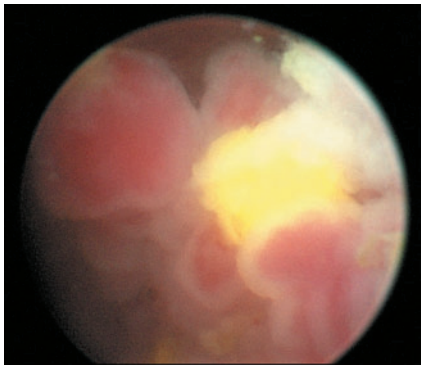


>>>1. A cystoscopy image of the dog's tumor revealing a polypoid mass and areas of necrosis and dystrophic mineralization.

serum chemistry profile, a urinalysis, an abdominal ultrasonographic examination, an abdominal and thoracic (three-view) radiographic examination, and a urine culture. The abdominal ultrasonographic examination showed a thickening of the bladder wall, with two masses measuring 0.38 x 0.64 cm and 1.9 x 2.3 cm and extending along the craniodorsal and cranioventral wall of the bladder. No other abnormalities were noted. Thoracic and abdominal radiographs revealed no abnormalities and no evidence of metastatic disease.

The dog was anesthetized and positioned in dorsal recumbency for a cystoscopic examination, which revealed multiple botryoid masses along the dorsal bladder wall close to the ureters (*Figure 1*). Histopathologic examination of biopsy samples revealed TCC, and a culture was positive for *Enterococcus* species that was susceptible to amoxicillin-clavulanic acid.

Treatment

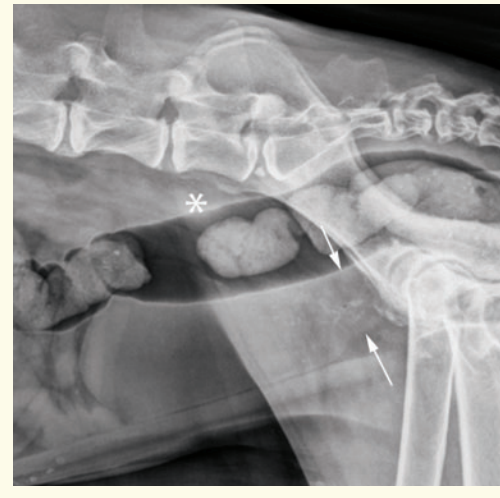
Surgical excision was not possible because of the proximity of the tumor to the ureters. Initial treatment included mitoxantrone chemotherapy administered intravenously every three weeks at a dose of 5 mg/m^2 in conjunction with piroxicam given orally daily at a dose of 0.3 mg/kg. A complete blood count was performed before and seven days after each mitoxantrone treat-

RADIOLOGY PERSPECTIVE

Silke Hecht, Dr. med. vet., DACVR, DECVDI

Diagnostic imaging in dogs with suspected bladder or urethral TCC is performed to identify lesions and, if identified, assess the extent of disease, evaluate for metastatic disease, and monitor tumor response to treatment. Survey radiographs of the abdomen are useful to identify enlarged sublumbar lymph nodes and evaluate the lumbar and sacrocaudal spine and the pelvis for evidence of osseous metastases or extension of the tumor into the spinal column or pelvis. The primary mass is usually not identified; however, dystrophic mineralization within the tumor or a thickened urethra may be seen in some cases (*Figure 2*).

Radiographic contrast procedures (cystography, urethrography) have largely been replaced by ultrasonography in the diagnosis of urinary tract neoplasia. However, lesions in an intrapelvic location may be better evaluated by using urethrography or computed tomography (CT) instead of ultrasonography in select cases.



>>>2. A lateral radiograph of the caudal abdomen of a 14-year-old Belgian shepherd with urinary bladder TCC. The radiograph shows irregular stippled mineralization in the plane of the caudal bladder and an unusual contour to the bladder neck area (*arrows*). Additional findings include mild sublumbar lymph node enlargement (*asterisk*) and incidental spinal degenerative changes.

It is important to note that 2D ultrasonography has historically been shown to be an inaccurate method of consistently measuring bladder masses.[17] Recently, however, 3D ultrasonography and CT have been shown to provide accurate bladder mass measurements, although the availability of CT and 3D ultrasonography is more limited than the availability of 2D ultrasonography.[18] On abdominal ultrasonographic examination, bladder TCCs

ment. Serum chemistry profiles were performed intermittently.

Follow-up

Treatment with mitoxantrone was continued every three weeks, and restaging tests were performed every six weeks to monitor tumor status. Restaging consisted of three-view thoracic radiographs, two-view abdominal radiographs, and abdominal ultrasonography to monitor primary tumor size as well as to monitor for evidence of the development of metastasis. For four months, the tumor remained stable based on imaging and clinical signs.

By the time the dog presented for its seventh mitoxantrone treatment, the owner noted that it was less active and was more particular about what it wanted to eat. A complete blood count, serum chemistry profile, urinalysis, and urine culture, as well as abdominal and thoracic imaging, revealed no explanation for the change in the dog's quality of life. No evidence of metastatic disease could be documented, and the tumor appeared relatively stable on abdominal ultrasonographic examination. The owner elected to discontinue chemotherapy since the dog's clinical signs were presumed to be from the chemotherapy treatment.

One month later, the dog presented to the primary care veterinarian because it was lethargic. Blood work revealed severe hyperglycemia, azotemia, and elevated liver enzyme activities. The owner elected to humanely euthanize at that time. The dog's survival time from diagnosis to death was five months. A necropsy was not performed to confirm the cause of the dog's declining quality of life.

Editors' note: *Two more perspectives—clinical pathology by Dr. Jennifer Scruggs and anatomic pathology by Dr. Jennifer Bernard—are available online at* **dvm360.com/TCC**.

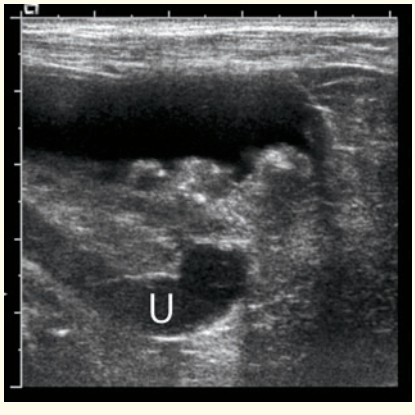
Dr. Scruggs Dr. Bernard

are usually sessile, irregularly shaped masses of variable echogenicity.[19] They are most commonly located at the level of the trigone but may be found in any location. Diffuse infiltration of the bladder wall instead of formation of a distinct mass is less common but possible. Urethral TCCs result in hypoechogenicity and irregular thickening of the urethral wall, often with a hyperechoic line along the epithelial surface.[20]

Possible concurrent findings include abnormalities of the prostatic gland, hydronephrosis or hydroureter secondary to ureteral obstruction (*Figure 3*), and evidence of metastatic disease to the lumbar spine, medial iliac lymph nodes, or other abdominal organs. Samples of bladder or urethral masses should ideally be obtained by means of ultrasound-guided traumatic catheterization ("suction biopsy") instead of percutaneous tissue sampling because of the risk of tumor cell implantation along the needle tract.[12] This is especially important for apical tumors in which surgery may be possible.

When monitoring a patient's response to treatment, it is important to keep the ultrasound technique and protocol (*e.g.* positioning, degree of bladder filling, radiologist) as consistent as possible between examinations to minimize the chance of measurement errors.

CT and magnetic resonance imaging (MRI) of the abdomen are useful in evaluating the primary tumor and associated changes. However, because of the need for general anesthesia, high cost, and low availability, the use of cross-sectional imaging in veterinary practice is currently limited. Thoracic imaging (radiography or, optimally, CT) should be routinely performed in patients with urinary tract TCC to evaluate for metastatic disease to the lungs or osseous structures.



>>>3. A sagittal ultrasonogram of the urinary bladder demonstrating an irregularly marginated, broad-based mass of mixed echogenicity with multiple strongly hyperechoic foci associated with the dorsal wall of the urinary bladder. A markedly dilated ureter (U) is seen dorsal to the bladder, consistent with ureteral obstruction by the mass.

MEDICAL PERSPECTIVE

Joe Bartges, DVM, PhD, DACVIM, DACVN, and Amanda Callens, BS, LVT

Dogs with TCC often have clinical signs of lower urinary tract disease (dysuria, stranguria, hematuria, and inappropriate urination) that may or may not be associated with concurrent bacterial UTIs. Alpha$_1$ antagonists may decrease lower urinary tract signs. Conventionally, prazosin (1 mg/15 kg body weight orally every 12 to 24 hours) and phenoxybenzamine (0.25 to 1 mg/kg body weight orally every eight to 24 hours) have been used; however, tamsulosin (0.001 to 0.1 mg/kg orally every 24 hours) has been shown to be superior.[21] Tamsulosin accumulates in urethral and prostatic tissues, providing a more sustained effect.[22,23]

Bacterial UTIs

Dogs with TCC are prone to bacterial UTIs, which are considered complicated because of urine retention due to mechanical blockage and presence of the cancer. Antimicrobial agents should be based on culture and sensitivity results since resistant UTIs are common. Although cystocentesis is controversial in dogs that have TCC due to the possibility of seeding tumors along the needle tract, urine should be collected by cystocentesis for culture and sensitivity testing. Not all bacterial UTIs require treatment; if clinical signs are not present or are no worse with a positive bacterial culture, then not treating is reasonable.[24]

Other nonantimicrobial treatments may be considered to aid in the control of bacterial UTIs, such as urinary antiseptics: methenamine mandelate (10 to 20 mg/kg orally with food every eight to 12 hours), methenamine hippurate (500 mg orally with food every 12 hours), or nitrofurantoin (3 to 4 mg/kg orally every 24 hours). Also consider probiotics, cranberry extract, and D-mannose.

Urethral obstruction

With time and despite treatment, urethral obstruction commonly occurs due to growth of the TCC at the urethral opening and down the urethral wall. Many patients still appear to feel well and do not have metastatic disease but are unable to urinate. There are several options for these obstructed patients—intermittent urinary catheterization, transurethral catheter placement, cystostomy catheter placement, and urethral stents. Intermittent urinary catheterization is only a temporary solution. If euthanasia is not considered, then alternative options for promoting urine voiding should be pursued.

Cystostomy catheters are low-profile tubes that are surgically placed into the urinary bladder and exit the abdominal cavity in the inguinal area. Owners empty the urinary bladder three to four times a day by using a syringe. We use bladder irrigation to help control bacterial UTIs with a cystostomy tube. Bladder irrigation is performed with a 0.02% chlorhexidine solution, and 20 to 30 ml is instilled in the bladder through the cystostomy tube after the urine is removed. It is allowed to dwell for 10 to 15 minutes, and then all but 10 ml is removed. This 10-ml 0.02% chlorhexidine solution is allowed to remain in the urinary bladder until the next time urine is removed.

Self-expanding nitinol urethral stents are placed by fluoroscopic or ultrasonographic guidance and permanently open the urethra. Most female dogs are incontinent after urethral stent placement, which may cause some concerns for home and owner exposure to chemotherapy if treatment is pursued after stent placement. Median survival times after stent placement are 32 to 78 days, with some dogs living up to a year.[25,26] Complications of stent placement include incontinence (39%), reobstruction (22%), stent migration, and bladder atony. About 20% of dogs with stents may develop urethral obstruction due to progressive growth of the tumor; of these about 10% obstruct due to ingrowth of the tumor through the stent, while about 90% obstruct due to growth of the tumor around the cranial and/or caudal aspects of the stent.[27]

Ureteral obstruction

If ureteral obstruction occurs due to a TCC, urinary diversion can be performed by either ureteral stents or a subcutaneous ureteral bypass device. Recently, laser ablation of TCC has been described, which may provide relief of clinical signs and possibly longer control of the cancer when used in conjunction with chemotherapy.[28]

SURGICAL PERSPECTIVE

Rachel Seibert, DVM

Complete TCC resection is difficult because masses usually involve the trigone region and are commonly multifocal, either within the bladder or in the bladder and urethra (in 56% of dogs in one study).[2] Because the important neurovascular structures of the bladder are located dorsally, resection of this area is likely to result in incontinence, ischemia, or both. In the aforementioned study of 67 dogs undergoing surgery for TCC, complete resection was possible in less than 3% of affected dogs.[2] For these reasons, surgical treatment is considered palliative in most cases and is often used in combination with chemotherapy and nonsteroidal anti-inflammatory drug (NSAID) therapy.

Partial cystectomy

Partial cystectomy is indicated to alleviate obstruction and for debulking in combination with chemotherapy. Major complications of partial cystectomy include dehiscence resulting in uroabdomen, incontinence if the neurovascular supply is damaged, pollakiuria due to reduced bladder capacity, incomplete tumor resection, and recurrence. Urinary catheters are recommended after cystectomy to keep the bladder decompressed, reducing tension on the suture line. In a study of 10 dogs undergoing partial cystectomy for TCC, nine of the dogs developed recurrence or metastasis, and 50% were euthanized within seven months of surgery.[29]

Although wide surgical margins can be attempted for apical masses, reduction in bladder capacity may result in persistent pollakiuria.[29,30] In people, more than 75% of the bladder can be excised with development of a near-normal capacity within three months[31]; however, the percentage of bladder that can be excised in dogs without long-term pollakiuria or incontinence is unknown. In dogs undergoing 90% cystectomy with only the trigone left intact, mean bladder capacity nine months after surgery was still decreased 72% from baseline, and all dogs had pollakiuria.[30] The rate of persistent pollakiuria is 20% when cystectomy includes 40% to 70% of the bladder.[29]

Because of metabolic complications and poor quality of life, complete cystectomy with urinary diversion is no longer performed.[32] An alternative to complete cystectomy is en bloc removal of the bladder neck and proximal urethra with preservation of the dorsal neurovascular pedicle. The goal of en bloc resection is to optimize the probability of achieving long-term tumor control, while attempting to preserve function. This technique was recently described in two dogs in which tumors were causing life-threatening obstruction of the urethra and ureters.[33] Both dogs regained continence by 17 days after surgery.

Surgical resection of TCC has been associated with tumor seeding of the abdominal wall or other distant sites.[34-36] Seeding of TCC is also suspected to occur with cystocentesis, traumatic urethral catheterization, cystoscopy, and laparoscopy.[12,36] When TCC is suspected, the surgeon should minimize tumor handling and change gloves and instruments before body wall closure to reduce the risk of iatrogenic spread.

Palliative treatment

Options for palliative treatment of trigonal TCC include placement of a permanent cystostomy tube, radiation therapy with or without placement of a self-expanding urethral stent, stent placement alone, or laser debulking. A permanent cystostomy tube permits urine diversion in patients that have urethral obstruction but patent ureters. Complications are reported in 49% of dogs and cats undergoing cystostomy catheter placement and include tube dislodgement or obstruction, urine leakage around the tube, persistent fistulation after tube removal, inflammation around the tube exit site, and death secondary to tube complications.[37] UTIs are also expected with long-term tube placement.

Tumors that obstruct the urethra and trigone can also be debulked with ultrasound-guided endoscopic diode laser ablation. When laser debulking is combined with medical management, the median survival time was 380 days in one report.[28] Multiple treatments are required for up to 50% of cases because of reobstruction with tumor or scar tissue. Other complications include stranguria, hematuria, urethral perforation, and spread of TCC within the urinary tract.[28]

MEDICAL ONCOLOGY PERSPECTIVE

Sara D. Allstadt, DVM, DACVIM (oncology)

Chemotherapy is often used to treat TCC because of the common trigone location of these tumors as well as common urethral and/or prostatic involvement. Reduction of tumor burden for TCC in dogs has not been well-studied but has been shown to prolong survival times.[38-40]

Chemotherapy agents

Systemic chemotherapy is often the primary form of treatment for TCC. Standard approaches include either treatment with NSAIDs alone or in combination with systemic chemotherapy agents. Treatment with NSAIDs such as piroxicam, deracoxib, and firocoxib has been reported, although piroxicam is commonly favored as the first-line NSAID by most oncologists, likely because of the higher-reported complete response rate to this drug.[41-43] Complete responses have been reported in a small number of cases treated with piroxicam alone, with a median survival time of 5.9 months.[41] In a report of 26 deracoxib-treated dogs, the median survival time reported was longer; however, those dogs received other chemotherapy agents after deracoxib treatments, likely influencing their overall survival times.[43]

Chemotherapy agents used in combination with NSAIDs include mitoxantrone, carboplatin, vinblastine, gemcitabine, vinorelbine, and metronomic chlorambucil. Intravesicular treatment with mitomycin C has also been reported.[44] While favorable tumor responses to cisplatin chemotherapy and piroxicam are noted, this combination is not recommended because of the high nephrotoxicity and risk of renal failure.[45,46] (*See Table 1 at* **dvm360.com/TCC** *for a summary of the study results for these chemotherapy agents.*)

Mitoxantrone combined with piroxicam is one of the more commonly used chemotherapy protocols for canine TCC.[47] The dose of mitoxantrone is typically 5 to 5.5 mg/m^2 administered intravenously every three weeks, and piroxicam is administered at 0.3 mg/kg orally once daily with food. Mitoxantrone can cause marked bone marrow suppression, so it is important to monitor the nadir via complete blood counts seven days after administration.

Piroxicam can cause both gastrointestinal upset and nephrotoxicosis, so routine monitoring of a patient's gastrointestinal signs and renal function is recommended. Omeprazole and famotidine are commonly used to reduce risk of gastrointestinal upset. Misoprostol is a synthetic prostaglandin E1 analogue commonly used to prevent NSAID-induced ulceration; however, in my experience, a high number of dogs experience gastrointestinal cramping and upset from this drug. It is also an abortifacient and, thus, women of child-bearing age should not be exposed to this drug.

Prognostic factors

Overall, the prognosis for dogs with TCC is poor, and most dogs succumb to this disease. But new treatments are being identified on a regular basis, and quality of life can be excellent in most dogs while receiving chemotherapy. Dogs with more advanced-stage TCC (*e.g.* larger tumors invading loco-regionally or with evidence of metastasis) have a less favorable prognosis than those with a lower-stage disease (*e.g.* smaller, more superficial tumors with no evidence of metastasis).[48] Younger dogs have an increased risk of metastasis to lymph nodes, dogs with prostate involvement have an increased risk of distant metastasis, and dogs with larger tumors have an increased risk for both nodal and distant metastasis.[49,50] Those with either loco-regional or distant metastasis, as well as dogs whose tumors invade the prostate, urethra, uterus, vagina, and pelvic canal, typically succumb to their disease or experience obstruction from their tumors quicker than those with smaller or more apically located tumors and without evidence of metastasis.[49,50]

The survival data for dogs with TCC is hard to summarize since many publications describe heavily pretreated dogs or dogs that go on to receive other treatments. However, some deductions may be made from the published data. With NSAID therapy alone, the expected median survival time is about six months, while adding chemotherapy to NSAID therapy is expected to increase the median survival time to approximately nine to 11 months (*see Table 1 at* **dvm360.com/TCC**). It is unknown at this time how new procedures such as urethral and ureteral stents and radiation therapy may affect survival times, but these modalities are expected to potentially increase survival.

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RADIATION ONCOLOGY PERSPECTIVE

Nathan Lee, DVM, DAVCR (radiation oncology)

Radiation therapy alone—or in combination with chemotherapy, surgery, or both—has been shown to be the best treatment in people with invasive TCCs, which are similar to canine TCC of the bladder.[51-55] Radiation therapy has not been extensively studied or recommended as treatment for canine TCC, most likely because of the relatively low availability of veterinary radiation centers. An early report looking at canine bladder cancers treated with intraoperative radiotherapy reported a high rate of post-radiation complications, with 46% of dogs experiencing increased frequency of urination, 46% urinary incontinence, 38% cystitis, and 15% stranguria.[56] The complications reported in that article were not surprising since the investigators treated the entire bladder and used a very high fraction dose of 21.88 to 27.00 Gy.[56] Even with the reported complications, the median survival time in that report was 16.8 months, which may indicate that radiation therapy may have a role in treatment of this tumor.[56]

A more recent report in 2004 investigated the use of piroxicam, mitoxantrone, and palliative radiation therapy (six weekly fractions of 5.75 Gy) in 10 dogs with TCC.[57] That study concluded that the combination of therapies, which has a response rate of 22% based on ultrasound measurements, was not superior to reports using mitoxantrone and piroxicam alone, which had a response rate of 34.5% based on ultrasound measurements.[46,57] Although this finding was not as promising as expected, 90% of patients had amelioration of their urinary clinical signs, and only three dogs were reported to have urinary incontinence as a late side effect.[57]

A recent study in 2012 investigated the use of radiation therapy to treat TCCs using intensity-modulated radiation therapy, along with image guidance in 21 dogs.[58] That study found that the combination of definitive radiotherapy (54 to 58 Gy delivered in 20 daily fractions on a Monday to Friday basis) and chemotherapy resulted in a median survival time of 654 days.[58] Acute urinary complications included hematuria (1) and stranguria (1).[58] Late complications reported were rectal (1), ureteral (1), and urethral (2) strictures.[58] Overall, 19% of patients developed grade three late gastrointestinal or genitourinary toxicosis that typically occurred six to 18 months after radiation therapy.[58] All late complications were successfully palliated with either stenting or surgery.[58]

At the University of Tennessee, we have successfully palliated patients with TCCs of the urinary bladder using 8-Gy fractions once a week for three treatments. With this therapy, we have seen clinical improvement with amelioration of urinary signs in most patients. Anecdotally, we see the most significant improvement seven to 14 days after radiotherapy. Palliative radiation therapy is typically reserved for patients that have failed chemotherapy options. The use of higher fraction sizes to treat TCCs was recently validated in a study from the University of Florida that determined the alpha/beta ratio of three established canine TCC cell lines was low, ranging from 2.67 to 4.[59] Cells with a low alpha/beta ratio tend to respond to large fraction sizes better than small fraction sizes, similar to late responding tissues in the body.

It is difficult to figure out the role of radiation therapy in the treatment of canine TCCs because of the few studies available, the small number of patients in each study, and the absence of practice standardization in veterinary radiation oncology. More study is required to fully define the role of radiation therapy in management of canine TCC of the bladder and determine how to reduce complications associated with this therapy. However, my experience combined with literature in both people and dogs affected by this disease suggest that this treatment modality should play a role in management of these dogs.

SUMMARY

At the University of Tennessee, piroxicam is the NSAID of choice for medical management of canine TCC, although deracoxib may be considered as well.[41,43] Mitoxantrone is typically chosen as the first-line chemotherapy drug, although other drugs considered and used in the treatment of canine TCC include vinblastine, carboplatin, and chlorambucil, based on the literature and clinical tolerance of canine patients.[45,47,60-63] It is important to note that several of these chemotherapy drugs should be dosed differently in small dogs (< 22 to 33 lb [10 to 15 kg]). Urethral and ureteral stenting, as well as radiation therapy, may all be considered as part of a management plan for TCC dogs.

For dogs being treated (particularly those treated with chemotherapy), we use imaging and restaging about every six to eight weeks to monitor response to treatment and determine if alternative therapy is indicated. VM

To view the references for this article, visit dvm360.com/TCC.

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